ANATOMICAL OBSERVATIONS ON THE BRAIN AND SEVERAL SENSE-ORGANS OF THE BLIND DEAF-MUTE, LAURA DEWEY BRIDGMAN.

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II.

I.— On the thickness and structure of the cerebral cortex.

PLATES III AND IV.

In a previous paper (AM. JOURN. OF PSYCHOLOGY, Vol. III, No. 3, Sept., 1890.) I have described some of the macroscopic features of the brain in question. I there stated the results of the measurements of the extent of the cortex (loc. cit. p. 336) as follows:

Extent of cortex, right hemisphere	= 98946.5 □ mm.
Extent of cortex, left hemisphere	= 101256.0 □ mm.
Total extent of cortex	= 200202.5 □ mm.

It has been recognized by all those who have studied the extent of the the cortex, that unless supplemented by observations on the thickness and character of the same, the figures for extent did not give a good ground for further inference. Jensen([45]) is, however, the only investigator who has up to this time made his studies thus complete.

It is, therefore, my purpose to report the results of the examination of the cortex of Laura Bridgman together with such conclusions as may be drawn from the results.

I.—The thickness of the cerebral cortex in general.

By way of preface I made a little excursion into the literature of the cortex to determine what was considered to be the normal thickness of that layer. It is highly probable that some of the work on this subject has escaped my notice, but what was found is tabulated (Table I.) with the purpose of showing how fully the various authors have stated the manner in which they obtained their results and what corrections had to be made, in certain cases, in order to have the results fairly comparable.

TABLE I.

Thickness of Cortex.

Date.	Authority.	Acquired defect.	No. of Brains Examined.	No. of localities in each hemisphere.	Special locality.	Average; localities not given.	Measuring Instruments.	Correction for Compasses.	Condition.	Correction for hardening.	Measurement taken in	Natural thickness of cortex in mm.	
1841	Parchappe[60]	. . . . .	. . .	.	Ant. lobes. Base & convexity	. .	. . . . .	. . .	. . . . .	. . .	. . . .	2.–3. 2.5–5.	Normal.
1841	Baillarger[54]	. . . . .	. . .	.	. . . . .	Average	. . . . .	. . .	Fresh (?)	. . .	Paris lines	3.37	
1865	Engel[53]	. . . . .	. . .	.	. . . . .	"	. . . . .	. . .	. . . . .	. . .	. . . .	2.2–3.7	
1875	Jensen[45]	. . . . .	4	18	. . . . .	. .	Compasses	+ 4 %	80 % alcohol	+ 2 %	. . . .	2.91	
1878	Richet[56]	. . . . .	. . .	.	. . . . .	"	. . . . .	. . .	Fresh (?)	. . .	. . . .	3.00	
1879	Bucknill and Tuke[57]	. . . . .	. . .	.	. . . . .	"	. . . . .	. . .	. . . . .	. . .	Inches	2.03	
1880	Danilewsky[49]	. . . . .	. . .	.	. . . . .	"	. . . . .	. . .	Fresh	. . .	. . . .	2.50	
1884	Conti[58.59]	. . . .	10 M.* 8 F.†	26	. . . . .	. .	Compasses	+ 4 %	Fresh	. . .	. . . .	2.25 M. 2.24 F.	
1886	Franceschi[61]	. . . .	10 M. 10 F.	35	. . . . .	. .	Compasses	+ 4 %	Fresh	. . .	. . . .	2.48 M. 2.46 F.	
1887	Luys[62]	. . . . .	. . .	.	. . . . .	Average	. . . . .	. . .	. . . . .	. . .	. . . .	3.–4.	
1888	Obersteiner[63]	. . . . .	. . .	Gyr. Post. Centr. Occipital Pole.		.	. . . . .	. . .	. . . . .	. . .	. . . .	4. 1.5	
1891	Donaldson	. . .	6 M. 3 F.	14	. . . .	. .	Micrometer Eye Piece	. .	Bichromate and alcohol	− 2 %	. . . .	2.92 M. 2.91 F.	
1872	Major[64]	Insanity	4	30	. . . . .	. .	Tephrylometer	. . .	80 % alcohol	. . .	Inches	2.37	
1875	Jensen[45]	" " Idiocy	2 M. 1 F. 2 F.	18	. . . .	. .	Compasess	+ 4 %	Fresh	+ 2 %	. . . .	2.68 M. 2.79 F. 2.48 F.	
1879	Bucknill and Tuke[57]	Insanity	33 M. 30 F.		. . . . .	Average	. . . . .	. . .	. . . . .	. . .	Inches	1.88 M. 1.85 F.	Defective.
1888	Cionini[65]	Gen'l Paralysis	8 M. 2 F.	31	. . . .	. .	Compasses	+ 4 %	Fresh	. . .	. . . .	1.83 M. 1.84 F.	
1891	Donaldson	Arrested develop'm't. L. B.	1	14	. . . .	. .	Micrometer Eye Piece	. .	Bichromate and alcohol	− 2 %	. . . .	2.59	

*M.= Male
†F.=Female

The authorities are arranged in chronological order, and in two groups: the first group containing the figures which apply to the cortex of normal persons, and the second the figures that apply to defectives. In this latter group I have only the measurements that apply to individuals with an acquired defect, as contrasted with those congenitally defective. The literature bearing on the cortex in these last has been brought together by Marchand([66]), and, though the facts are very interesting, they do not bear on our present problem and are therefore excluded.

The headings of the columns in Table I. will explain themselves, I trust, and the Table may be examined now without further explanation.

Omitting my own results, there are but six authors whose figures are of interest to us now. The manner in which the final figures in these cases have been obtained requires some explanation.

We desire to know the thickness of the cortex in its natural state, but the hardening reagents used for preserving the brain alter the thickness. In another place, I expect to make some general statements with regard to the weight and volume of nervous tissues as influenced by hardening reagents. Therefore I may state here only the results obtained, viz., that alcohol of 80% causes a decrease of 2% in the thickness of the cortex, while the bichromate and alcohol treatment (potassium bichromate $2\frac{1}{2}$% plus $\frac{1}{6}$ its volume of 95% alcohol for 6 to 8 weeks; washing in water for 24 hours; alcohol 95% for 2 days, and final preservation in 80% alcohol) causes an increase of 2%. As will be seen these corrections have been applied in Table I. Further, the manner of making the measurements has a very decided influence on the results. Direct experiment showed that the same localities measured with the compasses gave a thickness 4% less than when measured with a micrometer eye-piece under the microscope. There is no doubt in my mind that the microscopic method is the more accurate, hence I have corrected all the measurements made with compasses by the percentage above found.

There still remains the important question of the handling

of the figures for thickness after they are obtained. In general, the summit of a gyrus has the thickest cortex and the very bottom of the sulcus, the thinnest. In getting the thickness for any locality on the hemispheres at least two measurements, a maximum and minimum, are taken. Most investigators have measured the gyri at the points where the very thickest and very thinnest cortex was to be found, and for an average taken half the sum of these figures. The thinning of the cortex at the bottom of the sulci is, so to speak, sudden and excessive and the thinnest point deviates more from the intermediate cortex than does the thickest. Such being the case the resultant figure is somewhat too small. Conti([59]), Franceschi([61]) and Cionini([65]) give full tables and they have measured in the manner above described so that their averages represent one-half of the sum of the thickest and thinnest points in each gyrus. In the brains which I have examined the thickest portion was measured at the summit of the gyrus. The observations for the thinnest was taken at the side, about two-thirds of the distance from summit to sulcus. In making the average advantage was taken of the observation that one-third of the cortex lies at the summits of gyri and two-thirds is sunken in the sulci. The smaller figure was multiplied by 2, added to the larger figure and the sum divided by 3. As a consequence of this treatment I believe that my final average for the cortex of any particular gyrus is nearer the truth than it would be if half the sum of the thickest and thinnest points had alone been taken.

The figures which will be most useful to us can now be taken from Table I and presented in Table II, with the purpose of showing whether there is any difference in cortical thickness between males and females, or between the two hemispheres of the same brain; whether defectives correspond with normal persons; and what may be regarded as the normal thickness of the cortex.

Since the figures given in the Table II do not occur in their present form in the original tables of the authorities there quoted, I should perhaps add a word of explanation on the method by which they have been obtained.

Jensen([45]) gives a condensed statement for the normal brains, and in Table II his figures are simply corrected for the effect of alcohol and the use of compasses in measuring. His tables for the defectives are fuller and permit us to determine the averages for the two hemispheres. These are corrected in the manner above mentioned. In no case did he measure the cortex of the insula. Among the defectives one case which he gives is not entered in the table because it is that of a microcephalic.

Bucknill and Tuke([57]) give, without detail, the thickness of the normal cortex as .08 in. In a table of 63 pathological cases entered with great care and fullness, one column is devoted to the thickness of the cortex — also given without detail — in hundredths of an inch. This unit, approximately equal to .25 mm., is rather large when employed in so delicate a measurement. No statement as to the number, locality or method of their measurements is made. The cases were all adults.

Conti([59]) gives full tables. He claims twenty brains in his series. The measurements on two brains — females — are, however, so incomplete that they are not used here, hence he is credited with but eighteen brains in the table. Both hemispheres were not always examined. The total number of hemispheres represented in the table is only twenty-nine, 16 right and 13 left. His cases, principally adults, range in age from sixteen months to eighty years, but there is no evidence that the youngest cases should be excluded. Twenty-six localities in each hemisphere were measured but the cortex for the insula, if measured, is not specially recorded. In the prerolandic and post-rolandic regions only the summits of the gyri and the depths of the sulci were measured. In the rolandic region intermediate measurements on each wall of the gyri were taken. The averages were obtained by summing and dividing the figures as they stand in his tables and then correcting the final results for the use of compasses. The original measurements were made in tenths of a millimeter.

Franceschi([61]) gives full and very complete tables. He examined the cortex at 35 localities on both hemispheres of twenty brains, principally from adults of advanced age, 10

males and 10 females. The measurements taken in tenths of a millimeter, and were made at the summits of the gyri and the depths of sulci. The cortex of the insula was included. The figures in Table II. are obtained directly from those of his tables, save that they have been corrected for use of compasses.

Major([64]) tested the thickness of the cortex at thirty localities on both hemispheres of the brains of four adult insane patients, the sex not given. For each locality he gives only the mean depth using one-fifth of an inch as his unit of measure. This unit is, of course, too large. He measured the insular cortex. His figures for the cortical thickness give the mean depth without detail as to the method of obtaining the mean. The instrument used, the tephrylometer, consisted of a thin walled graduated glass tube. This was pressed into the brain substance at any desired point, then, the upper end being closed by the finger, withdrawn, when a plug of brain substance remained within the tube and on this plug the thickness of the cortex is read off by the aid of the scale etched in the tube. The figures in Table. II are the simple averages of those in his tables without any corrections. Concerning the accuracy of this method of measuring the cortex there are no observations.

Cionini([65]) presents his results from the examination of fifteen adult brains, ten males, five females, all cases of general paralysis. The number of localities was 31, but in other respects the details are similar to those in the case of Conti. It occurs, however, that in five cases, three males and two females, the tables are so incomplete that they cannot be used for averages, and hence only ten cases are represented. The figures in Table II. are obtained as in the case of Conti.

A glance at Table II. shows that in both normals and defectives the average thickness is very slightly, —.01 to —.04mm., greater in the males in five out of the six cases (larger number underlined). There is a slightly greater difference between the two hemispheres, which is in favor of the left hemisphere as the figures stand (eight out of thirteen cases). In discussing the absolute thickness of the cortex as reported we have, of course, to throw out the defectives, who are, *ipso facto*, expected to have a thinner cortex.

TABLE II.

Thickness of Cortex.

	MALES.					FEMALES.					
Authority.	No. of Brains.	Defect.	Right Hemisph'e.	Left Hemisph'e.	Average.	No. of Brains.	Defect.	Right Hemisph'e.	Left Hemisph'e.	Average.	
Jensen[45]	4	. . . .	. . . .	. . . .	2.91	. . .	. . . .	. . . .	. . . .	. . . .	
Bucknill and Tuke[57]	. . .	. . . .	. . . .	. . . .	2.03	. . .	. . . .	. . . .	. . . .	. . . .	
Conti[58-59]	10	. . . .	*2.29	2.21	†2.25	8	. . . .	2.24	2.25	2.24	
Franceschi[61]	10	. . . .	2.479	2.474	2.48	10	. . . .	2.463	2.457	2.46	Normal.
Donaldson	6	. . . .	2.91	2.94	2.92	3	. . . .	2.89	2.92	2.91	
Major[64]	‡4	Insanity	2.368	2.379	2.37	. . .	. . . .	. . . .	. . . .	. . . .	
Jensen[45]	2	Insanity	2.68	2.68	2.68	1	Insanity	2.809	2.777	2.79	
	. . .	. . . .	. . . .	. . . .	. . . .	2	Insane Idiots	2.46	2.50	2.48	
Bucknill and Tuke[67]	33	Insanity	. . . .	. . . .	1.88	30	Insanity	. . . .	. . . .	1.85	Defective.
Cionini[65]	8	General Paralysis	1.809	1.851	1.83	2	General Paralysis	1.778	1.809	1.79	
Donaldson	. . .	. . . .	. . . .	. . . .	. . . .	1	Arrested development	2.55	2.62	2.59	

* Where there are averages for the two hemispheres the larger figure is doubly underlined.
† Where there are averages for the two sexes the larger figure is underlined.
‡ Sex not given.

At the moment I have no explanation to offer of the various figures given for the absolute thickness in normal persons and will simply point out that my figures agree most closely with those of Jensen.

It appears, therefore, that the average thickness for the two sexes is nearly alike, what difference there is being in favor of the males; that the left hemisphere more often has the thicker cortex; that in defectives (not congenital) it is thinner than in normal persons, and that the figures given for the absolute thickness in normal persons are at present irreconcilable. With this I conclude the introductory study of the subject.

II. *Comparison of the cortex of Laura Bridgman with that of nine normal brains (six males; three females).*

The normal brains were obtained in New York about a year ago, and I am indebted to the courtesy of several medical gentlemen of the city for them. There is no reason to think that any of these specimens were from persons of more than average intelligence, hence on that score they are comparable with the Bridgman brain. They were hardened in the same manner that the latter was (*vide* p. 9). Samples of cortex were taken in all cases from 14 localities on each hemisphere, each locality being designated by an arbitrary number.

Plate III shows the localities with the numbers used, and is intended to take the place of a written description.

In Table III. I give the cortical areas in which the localities are situated.

All the samples from the several localities were treated in the same manner, viz.: imbedded in celloidin, cut in sections about 0.1 mm. thick and measured, unstained, under a low magnifying power. It is hardly necessary to add that all the

TABLE III.

Locality.	Cortical Area for.	Locality.	Cortical Area for.
1.	Speech motor ?	8.	Sight, sensory.
2.	Speech, motor.	9.	— ?
3.	Speech?	10.	Taste and smell, sensory.
4.	Head and eyes, motor.	11.	Sight, sensory.
5.	Arm, motor.	12.	Touch, sensory.
6.	Hearing, sensory.	13.	Leg, motor.
7.	— ?	14.	Sight, sensory.

measurements were concluded before any calculations were begun and that precaution was taken to keep the results unprejudiced.

Figures for the average thickness at each locality having been obtained from all the brains in the manner above described, the localities were arranged in order, from the thickest to the thinnest, and the tables thus formed were plotted as curves. *Vide* Plate IV.

The principal results are tabulated in Table II (under Donaldson, normals), and in Table IV a further analysis is given. The figures for males and females being separated in Table IV, those for the right and left hemispheres are given in each group and the individuals in each group are ranged according to age. This last arrangement was made to see whether they showed a decrease in cortical thickness with advancing age. Conti[58] reports that the cortex decreases regularly from a maximum at 3 years to a minimum in extreme age. I do not pretend to discuss the question here but simply refer to the table to show that these brains when thus arranged do not exhibit a decrease.

TABLE IV.

Thickness of Cortex in Controls and in Laura Bridgman.

MALES. Arranged according to age.				FEMALES. Arranged according to age.			
Age.	Weight in grms.	R. H.	L. H.	Age.	Weight in grms.	R. H.	L. H.
35	1419	* 2.81	2.81	40	1196	2.74	2.74
35	1443	2.87	3.09	45	1173	2.80	3.00
39	1393	2.77	2.86	Adult	1312	3.12	3.02
45	1367	2.90	2.93				
57	1464	2.96	2.91				
Adult	1210	3.14	3.07				
		2.91	2.94			2.89	2.92
General Average, 2.92.				General Average, 2.90.			
Laura Bridgman,				60	1204	2.55	2.62
				General Average, 2.59			

* The underlining has the same significance as in Table II.

The cortex of the left hemisphere is in five cases the thicker, while that of the right is so in four. The maximum difference between the two hemispheres of the same individual is .22 mm. (2.87 to 3.09). The averages for the males and females are nearly alike, the males being a trifle, .02 mm., thicker.

If the results for each locality are averaged for all the controls, these averages arranged in a series from the largest to the smallest and this series plotted as a curve, then the curve has the form indicated by the continuous black ink line on Plate IV. In that curve the insula, as pointed out by Major (64), has the thickest cortex. Next follows the convex surface of the hemispheres with little variation, and then the thickness gradually decreases in the mesal, occipital and orbital cortex, in the order named. Table V gives the figures from which this curve is formed as well as the figures for the two component curves, viz.: that for the males and that for the females.

TABLE V.

Averages for each locality. All controls, I.
Averages for each locality. Controls, Male, II.
Averages for each locality. Controls, Female, III.
Unit of measure, 1 mm.

Locality.	I. Average for all Controls.	II. Average for Controls, (6) Male.	III. Average for Controls, (3) Female.
3	3.38	3.48	3.33
7	3.15	3.02	3.43
6	3.10	3.05	3.18
4	3.09	3.12	3.04
2	3.08	3.06	3.12
5	3.08	3.10	3.04
10	3.04	3.03	3.06
1	2.98	2.92	3.06
13	2.86	2.82	2.94
12	2.75	2.83	2.60
11	2.65	2.65	2.66
8	2.61	2.67	2.50
9	2.53	2.60	2.41
14	2.52	2.59	2.38
	Average,	2.92	2.91

By these figures I aim to show the normal thickness of the cortex at the given localities.

The figures which form the basis for the curve of the Bridgman brain are given in Table VI. The average thickness of this cortex (see Table IV) is 2.59 mm., which is 0.32 mm. below the average for all the females and 0.15 mm. below that for the female in whom the cortex was thinnest.

TABLE VI.

I. Averages of the several localities. L. B., right hemisphere.
II. Averages of the several localities. L. B., left hemisphere.
III. Averages of the several localities. L. B., both hemispheres.

LAURA BRIDGMAN.

Locality.	I. R. H.	II. L. H.	III. Average.
3	3.45	2.98	3.22
7	2.93	2.72	2.83
6	2.26	2.56	2.41
4	2.98	2.77	2.88
2	2.74	2.89	2.82
5	2.61	2.75	2.68
10	2.51	2.41	2.46
1	2.70	2.54	2.62
13	2.81	2.69	2.75
12	2.70	2.56	2.63
11	1.99	2.72	2.36
8	2.16	2.48	2.32
9	1.99	2.27	2.13
14	1.92	2.35	2.14
	Aver. 2.55	Aver. 2.62	Aver. 2.59

The curves for the Bridgman figures are plotted on Plate IV. That for the left hemisphere is indicated by a broken line (dashes), and that for the right hemisphere by the line of long and short dashes. Attending for the moment to these we observe a remarkable drop at 6; from 4 to 12 both curves are generally low with a special depression at 10, and from 12 to the end they run at different levels.

It will be seen at a glance that these two curves are fairly accordant until locality 11 is reached. Here they are widely divergent, approach somewhat at 8, again to diverge at 14.

Taking up the peculiarities of the Bridgman cortex then in the order in which they occur we find the insula (3) thinner on the left side. Both sides very thin at 6, the auditory area. Locality 2, the area for motor speech, is well developed on both sides. From 4 to 13 the development is poor, specially so at 10, area for taste and smell. At 12, the area for dermal sensations, the curve is high again, and from that point on commences the remarkable divergence in the curves of the two hemispheres, that for the left side being much higher at 11, 8 and 14, all of which are within the visual area.

Referring now to the description which I have previously given (op. cit.) of the macroscopic features of this brain, I may briefly attempt to collate them with the measurements of the cortex.

The insula (3) on the left side was found less well developed. It has the thinner cortex. *Vide* Waldschmidt([67]).

At the auditory area (6) I could not decide on any macroscopic defect, but have since determmined that the first temporal gyrus at its caudal end, especially on the right side, was abnormally slender. The cortex is decidedly thin on both sides, most markedly so on the right. At the area for motor speech, the left side showed a clear lack of development (depression), but the cortex was not particularly thin for this brain.

At 10, the area for taste and smell, there was a general lack of development, exhibited by the entire temporal lobe. This is easily explained by the slow growth of this portion of the brain, a growth which was quite incomplete at the period when Laura was taken ill (2 years). The glossopharyngeal nerves appeared normal, but the olfactory bulbs and tracts were small, though not so small as in the case of some normal persons. The thinness of the cortex at this point (10) appears therefore as a part of the general arrest in growth.

Passing now to the visual area it was noticed macroscopically that both occipital lobes were blunted, but the right side turned out in every way to be much the more defective and anomalous. Concordantly the cortex of this right side at 11, 8, and 14 is much thinner than that of the left.

It must be recalled here that although at the age of two years, Laura became completely blind in her left eye, yet she retained some remnant of vision with her right eye up to her eighth year. This has left its mark on the entire central apparatus for vision. The right optic nerve is larger than the left.

Area of cross-section of R. optic nerve = 5.00 □ mm.
" " " " " L. " " = 3.38 " "

The relation in the tracts is, of course, reversed:

Area of cross-section of R. optic tract = 3.13 □ mm.
" " " " " L. " " = 4.69 " "

On the one hand then we have loss of vision in left eye at 2 years of age, associated with the smaller optic nerve and tract—a defectively developed right occipital lobe and a thin cortex in the right visual area. On the other hand we have some vision in the right eye up to the eighth year of age, associated with the larger optic nerve and tract, the more normal occipital lobe and the thicker cortex.

The general thinning of the motor cortex I would explain in part by the absence of the fibres through which the motor areas are normally associated with the sensory areas—here defective —and in part by the smaller size of some of the cell elements and non-development of others, resulting from lack of stimuli. The defects in the visual and auditory area follow directly from the loss of the corresponding sense organs and consequent arrest of growth. When the loss is not at first complete a good deal of subsequent development is possible. Why the speech-centre has not a thinner cortex I cannot, at the moment, explain.

In considering the fact that the sensory centers are much more affected than the motor, it should be remembered that aside from the special loss due to arrest and possibly degeneration falling less on the motor than on the sensory centres, there is the physiological difference that each motor centres can be excited by way of any sensory centre, and hence, so long as any senses are left, the motor centres must be stimulated to some degree, while the destruction of the special sense-organ throws a given sensory centre quite out of function. The physiological conditions in the two cases are therefore quite different and in favor of the development of the motor side.

For reference. I introduce here several tables containing the details of the figures just given.

Table VII. gives the maximum and minimum thickness of the cortex as observed at each locality on Laura Bridgman and the nine controls. The maximum was taken at the summit of the gyrus and the minimum at the side — not at the bottom of the sulcus. The average of the maximum and minimum is obtained by doubling the minimum, adding the result to the maximum and dividing the sum by three. This average figure is given in the third column for each hemisphere. The averages at the foot of the first and second columns are obtained by dividing the sum of these columns by fourteen. All the figures in this table are corrected for hardening, so that they represent the natural thickness of the cortex. The observations for the males and females are separated.

TABLE VII.

Females.

Specimen.	BRIDGMAN.						I.						VI.						XI.					
Locality.	R. H.			L. H.			R. H.			L. H.			R. H.			L. H.			R. H.			L. H.		
	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.
1	2.91	2.59	2.70	3.49	2.07	2.54	2.91	2.59	2.70	3.43	3.11	3.22	3.17	2.98	3.04	3.75	3 56	3.62	3.36	2.85	3.02	3.17	2.59	2.78
2	3.30	2.46	2.74	3.23	2.72	2.89	3.56	3.24	3.35	3.36	2.72	2.93	3.88	2.91	3.23	3.24	2.91	3.02	3.24	2.79	2.94	3.56	3.11	3.26
3	3.88	3.24	3.45	3.11	2.91	2.98	3.88	2.91	3.27	3.88	3.24	3.45	3.88	3.30	3.49	3.75	3.43	3.54	3.49	2.26	2.67	3.56	2.33	2.74
4	3.24	2.85	2.98	3.40	2.46	2.77	2.98	2.39	2.59	3.69	3.24	3.39	3.43	3.24	3.30	3.24	2.91	3.02	3.11	2.52	2.72	3.36	3.11	3.19
5	3.24	2.30	2.61	3.07	2.59	2.75	3.30	2.81	2.97	3.79	3.33	3.48	3.72	2.91	3 20	3.56	3.07	3.23	2.52	2.20	2.31	3.36	2.91	3.06
6	2.91	1.94	2.26	2.91	2.39	2.56	3.62	3.36	3.45	3 75	3.24	3.41	3.88	3.49	3.62	3.30	2.59	2.83	3.36	2.98	3.11	3.49	2.26	2.67
7	3.24	2.78	2.93	2.98	2.59	2.72	3.56	3.17	3.30	3.88	3.69	3.75	3.56	3.24	3.35	3.56	3.24	3.35	3.56	3.36	3.43	3.43	3.36	3.38
8	2.59	1.94	2.16	3.04	2.20	2.48	2.91	1.94	2.26	2.91	2.01	2.31	3.17	2.98	3.04	3.24	2.39	2.67	2.72	2.26	2.41	2.39	2.26	2.30
9	2.20	1.88	1.99	2.52	2.14	2.27	2.72	1.94	2.20	3.56	2.26	2.69	3.24	2.39	2.67	2.78	2.07	2.31	2.59	1.94	2.16	2.78	2.26	2.43
10	2.84	2.35	2.51	2.75	2.24	2.41	2.85	2.14	2.38	2.59	2.26	2.37	2.88	2.91	3.23	3.75	3.11	3.32	3.88	3.56	3.67	3.88	3.11	3.37
11	2.20	1.88	1.99	2.98	2.59	2.72	3.04	2.91	2.95	3 88	2.39	2.89	3.33	2.56	2.82	3.07	2.59	2.75	2.65	1.94	2.18	2.65	2.20	2.35
12	2.91	2.59	2.70	2.65	2.52	2.56	3.11	2.07	2.42	2.91	2.65	2.74	3.75	2.52	2.93				2.91	2.39	2.56	2.59	2.20	2.33
13	3.24	2.59	2.81	3.04	2.52	2.69	3.43	3.11	3.22	3.43	2.59	2.87	3.24	2.98	3.07	3.36	2.98	3.11	3 30	2.72	2.91	2.78	2.26	2.43
14	2.14	1.81	1.92	2.91	2.07	2.35	2.59	1.94	2 16	2.72	2.39	2.50	3.04	2.59	2.74	2.72	2.33	2.46	2.78	2.07	2.31	2.46	1.94	2.11
Aver.	2.92	2.37	2.55	3.01	2.43	2.62	3.18	2 61	2.80	3.41	2.79	3.00	3.51	2.93	3.12	3.33	2.86	3.02	3.11	2.56	2.74	3 10	2.58	2.74

TABLE VII.—*Males.*

Specimen	II.						III.						IV.					
Locality.	R. H.			L. H.			R. H.			L. H.			R. H.			L. H.		
	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.
1	3.04	2.65	2.78	3.30	2 20	2.57				3.24	2.91	3.02						
2	3.36	2.91	3.06	3.56	3.11	3.26	3.24	3.11	3.15	3.11	2.98	3.02	3.49	2.91	3.10	3.24	3.17	3.19
3	3.56	2.91	3.13	3.88	3.88	3.88	3.88	2.91	3.23	3.56	2.91	3.13	3.24	2.75	2.91	3.88	3.36	3.53
4	3.24	3.17	3.19	3.24	2.98	3.07	3.11	2.72	2.85	3.24	2.59	2.81	3.81	3.56	3.64	3.69	3.24	2.39
5	3.56	2.91	3.13	3.40	2.73	2.95	3.30	2.75	2.93	3.36	2.65	2.89	3.56	3.30	3.39	3.56	3.24	3.35
6	3.56	3.11	3.26	3.56	3.11	3.26	3.56	3.24	3.35	3.24	2.91	3.02	3.88	3.17	3.41	3.56	2.91	3.13
7	3.11	2.91	2.98	3.43	2.39	2.74	2.91	2.59	2.70	3.36	3.24	3.28	3.88	3.24	3.45	3.56	3.43	3.47
8	2.78	2.59	2.65	2.59	2.20	2.33	2.59	2.26	2.37	2 91	2.59	2.70	3.17	2.91	3.00	2.85	2.59	2.68
9	2.91	1.94	2.26	2.65	2.39	2.48	2.59	2.20	2.33	2.72	2.01	2.25	3.24	2.91	3.02	3.24	2.72	2.89
10	3.11	2.91	2.98	3.56	2.78	3.04	3.88	3.24	3.45	3.24	3.24	3.24	3.88	3.56	3.67	3.56	2.91	3.13
11	3.56	3.11	3.26	3.30	2.65	2.87	2.39	1.94	2.09	2.91	2.65	2.74	3.24	2.71	2.89	2.58	2.07	2.24
12	3.36	2.26	2.63	3 36	3.11	3.19	3.24	2.59	2.81	2.85	2.39	2.54	3.24	2.59	2.81	3.49	3.24	3.32
13	3.36	2.59	2.85	3.36	2.59	2.85	2.91	2.26	2.48	2.65	2.52	2.56	3.24	2.85	2.98			
14	2.59	2.39	2.46	2.65	2.46	2.52	2 52	2.07	2.22	2.91	2.72	2.78	3.04	2.39	2.61	2.59	2.39	2.46
Aver.	3.22	2.74	2.90	3.27	2.76	2.93	3.09	2.61	2.77	3.09	2.74	2.86	3.45	2.99	3.14	3.32	2.94	3.07

TABLE VII.—*Males.*

Specimen	IX.						X.						XII.					
Locality.	R. H.			L. H.			R. H.			L. H.			R. H.			L. H.		
	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver	Max.	Min.	Aver.
1	3.56	2.80	3.05	3.24	2.33	2.63	2.98	2.78	2.85	3.49	3.30	3.36	3.36	2.91	3.06			
2	3.69	3.36	3.47	3.36	2.98	3.11	2.59	2.26	2.37	3.11	2.65	2.80	3.24	2.78	2.93	3.56	3.11	3.26
3	4.01	3 88	3.92	3.88	3.49	3.62	3.88	3.56	3.63	3.88	3.56	3.67	3.88	3.56	3.66	3.56	3.36	3.43
4	3.17	2.91	3.00	3.36	2.91	3.06	3.49	3.17	3.28	3.43	3.24	3.30	3.11	2.59	2.76	3.56	2.78	3.04
5	3.23	2.98	3.06	3.49	2.91	3.10	3.24	2.91	3.02	3.88	3.43	3.58	3.56	2.91	3.13	3.24	2.39	2.67
6	3.56	2.33	2.74	3.24	3.17	3.19	3.11	2.72	2.85	2.72	2.59	2.63	3.49	3.11	3.24	3.17	2.26	2.56
7	3.88	2.91	3 23	3.36	2.72	2.93	3.56	2.65	2.95	3.69	2.52	2.91	2 59	2.39	2.46	3.36	2.98	3.11
8	3.24	2.65	2.85	3.56	2.26	2.69	2.98	2.52	2.67	3.30	2.85	3.00	2.78	2.33	2.48	2.91	2.46	2.61
9	2.91	2.59	2.70	2.85	2.46	2.59	2.72	2.59	2.63	2.85	3.34	3.18	2.72	2.39	2.50	2.59	2.20	2.33
10	3.24	2.65	2.85	2.91	2.39	2 56	2.59	2.39	2.46	3.88	3.30	3.49	3.24	2.59	2.81	3.36	2.47	2.73
11	2.46	2.39	2.41	3.11	2.39	2.63	3.36	2.59	2.85	3.43	2.59	2.87	3.11	2.26	2.54	2.59	2.33	2.42
12	3.30	2.59	2.83	3.36	2.52	2.80	2.65	2.46	2.52	3.36	2.78	2.97	3.36	2.26	2.63	3.43	2.59	2.87
13	3.30	2.72	2.91	3.56	3.24	3.35	3.11	2.98	3.02	3.49	2.24	2.66	3.88	2.33	2.85	2.72	2.46	2.55
14	2.91	2.14	2.40	2.59	2.39	2.46	3.11	2.98	3.02	3.04	2.72	2.83	2.59	2.26	2.37	3.56	2.72	3.00
Aver.	3.32	2.78	2.96	3.28	2.73	2.91	3.10	2.75	2.87	3.40	2.94	3.09	3.21	2.62	2 81	3.20	2.62	2 81

Table VIII. is derived from Table VII. by arranging the figures for the average thickness of each locality in each hemisphere in vertical columns, and getting the averages of these for the females alone, for the males alone, and for both together.

TABLE VIII. *Controls Only.*

Locality.	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.		
	2.70	3.35	3.27	2.59	2.97	3.45	3.30	2.26	2.20	2.38	2.95	2.42	3.22	2.16	I R.	Females.
	3.22	2.93	3.45	3.39	3.48	3.41	3.75	2.31	2.69	2.37	2.89	2.74	2.87	2.50	I L.	
	3.04	3.23	3.49	3.30	3.20	3.62	3.35	3.04	2.67	3.23	2.82	2.93	3.07	2.74	VI R.	
	3.62	3.02	3.54	3.02	3.23	2.83	3.35	2.67	2.31	3.32	2.75		3.11	2.46	VI L.	
	3.02	2.94	2.67	2.72	2.31	3.11	3.43	2.41	2.16	3.67	2.18	2.56	2.91	2.31	XI R.	
	2.78	3.26	2.74	3.19	3.06	2.67	3.38	2.30	2.43	3.37	2.35	2.33	2.43	2.11	XI L.	
Average: Females.	3.06	3.12	3.33	3.04	3.04	3.18	3.43	2.50	2.41	3.06	2.66	2.60	2.94	2.38		
	2.78	3.06	3.13	3.19	3.13	3.26	2.98	2.65	2.26	2.98	3.26	2.63	2.85	2.46	II R.	Males.
	2.57	3.26	3.88	3.07	2.95	3.26	2.74	2.33	2.48	3.04	2.87	3.19	2.85	2.52	II L.	
		3.15	3.23	2.85	2.93	3.35	2.70	2.37	2.33	3.45	2.09	2.81	2.48	2.22	III R.	
	3.02	3.02	3.13	2.81	2.89	3.02	3.28	2.70	2.25	3.24	2.74	2.54	2.56	2.78	III L.	
		3.10	2.91	3.64	3.39	3.41	3.45	3.00	3.02	3.67	2.89	2.81	2.98	2.61	IV R.	
		3.19	3.53	3.39	3.35	3.13	3.47	2 68	2.89	3.13	2.24	3.32		2.46	IV L.	
	3.05	3.47	3.92	3.00	3.06	2.74	3.23	2.85	2.70	2.85	2.41	2.83	2.91	2.40	IX R.	
	2.63	3.11	3.62	3.06	3.10	3.19	2.93	2.69	2.59	2.56	2.63	2.80	3.35	2.46	IX L.	
	2.85	2.37	3.63	3.28	3.02	2.85	2.95	2.67	2.63	2.46	2.85	2.52	3.02	3.02	X R.	
	3.36	2.80	3.67	3.30	3.58	2.63	2.91	3.00	3.18	3.49	2.87	2.97	2.66	2.83	X L.	
	3.06	2.93	3.66	2.76	3.13	3.24	2.46	2.48	2.50	2.81	2.54	2.63	2.85	2.37	XII R.	
		3.26	3.43	3.04	2.67	2.56	3.11	2.61	2.33	2.73	2.42	2.87	2.55	3.00	XII L.	
Average: Males.	2.92	3.06	3.48	3.12	3.10	3.05	3.02	2.67	2.60	3.03	2 65	2.83	2.82	2.59		
Average: Males and Females.	2.98	3.08	3.38	3.09	3.08	3.10	3.15	2.61	2.53	3.04	2 65	2.75	2.86	2.52		

Table IX. gives the difference in the thickness of the cortex in the two hemispheres of those controls in which the difference is greatest. The figures on which this table is based are found in the "average" columns of Table VII. The controls are grouped into males and females and the instance of greatest dlfference found for each group. To be compared with this is the difference in the same localities in the Bridgman brain. The figures for fhe latter show that the differences are much within the extremes of the controls

except at those localities where the largest difference is to be expected i. e., 3, 8, 11, 14—where they may exceed those of the controls. The roman numeral indicates the number of the specimen and the side which is larger is first designated, so that VI L.-VI R. means that the left hemisphere has the thicker cortex in control VI. It is not without interest in this case that among the females, 9, and among the males, 11 out of the 14 cases have the left cortex the thicker.

TABLE IX.

Greatest Differences in Cortical Thickness.

	FEMALES.		MALES.		L. B.	
Loc.	Gr. Diff.	Specimen.	Gr. Diff.	Specimen.	Gr. Diff.	
1.	.58	VI L.—VI R.	.51	X L.—X R.	.16	R. L.
2.	.42	I R.—I L.	.43	X L.—X R.	.15	L. R.
3.	.18	I L.—I R.	.75	II L.—II R.	.47	R. L.
4.	.80	I L.—I R.	.28	XII L.—XII R.	.21	R. L.
5.	.75	XI L.—XI R.	.56	X L.—X R.	.14	L. R.
6.	.79	VI R.—VI L.	.68	XII R.—XII L.	.30	L. R.
7.	.45	I L.—I R.	.65	XII L.—XII R.	.21	R. L.
8.	.37	VI R.—VI L.	.33	X L.—X R. III L.—III R.	.32	L. R.
9.	.49	I L.—I R.	.55	X L.—X R.	.28	L. R.
10.	.30	XI R.—XI L.	.54	IV R.—IV L.	.10	R. L.
11.	.17	XI L.—XI R.	.65	III L.—III R. IV R.—IV L.	.73	L. R.
12.	.32	I L.—I R.	.56	II L.—II R.	.14	R. L.
13.	.48	XI R.—XI L.	.44	IX L.—IX R.	.12	R. L.
14.	.34	I L.—I R.	.63	XII L.—XII R.	.43	L. R.

III.—Histological Examination.

The Bridgman brain was not well enough preserved to admit of a very fine microscopical examination. Some points can be made out, however, on sections .02 mm. thick, stained with hæmatoxylin and eosin, or hæmatoxylin and carminic acid, or with Weigert-Pal hæmatoxylin. Whatever general statements are made are always in comparison with the nine controls, from which sections were also cut and similarly stained.

The cells generally in the Bridgman cortex have abundant pigment—the nuclei often somewhat irregular and the nucleoli sometimes single and clear, often multiple and unclear, and, at times, wanting. Where the cortical granules form layers they appear abundant, as a rule, and immature (i. e., without

angles), as though they had been arrested in their growth. The general impression one gets is, that the large nerve cells are neither so large nor so numerous as in the normal brains. Of cell processes and abundance of fibres one can only say, that there appear less of both in all localities, and hasten to add, that the poor condition of the material makes itself painfully felt at this point.

It seemed worth while, however, to select sections from several localities, especially those in which the cortex of the Bridgman brain appeared thin, and attempt to get some notion of the development of the cell elements at these points.

To arrive at this result I counted the number of cells above a given diameter in a strip of the cortex, comparing the number found in the Bridgman cortex with that in two controls. For results see Table X.

TABLE X.

To show the average number of cells 12 μ in transverse diameter which occur in 0.01 □ mm. of cerebral cortex at the localities named. Sections .02 mm. thick.

	MALE.			FEMALE.					
	Control III.			Control XI.			Laura Bridgman.		
Locality.	*R.	†L.	Aver.	R.	L.	Aver.	R.	L.	Aver.
Speech, 2	.85	1.10	0.975	1.06	1.16	1.11	.93	.80	0.865
Insula, 3	1.15	1.04	1.10	1.15	1.03	1.09	1.00	1.07	1.035
Head and Eyes, 4	1.13	1.40	1.26	1.03	1.46	1.25	1.11	1.19	1.15
Hearing, 6	1.23	.99	1.11	1.23	1.21	1.225	.81 c	.92	0.865
Taste and Smell, 10	.82	1.12	0.97	1.34	.97	1.155	.86	1.05	0.955
Sight, 11	1.03	.99	1.01	1.08	.95	1.015	.47 c	1.01	0.74
Sight, 14	1.13	1.08	1.105	.99	1.08	1.03	.40 c	.92	0.66
Average,			1.075			1.125			0.895

* R. = Right hemisphere.

† L. = Left hemisphere.

To obtain these figures the following method was employed. The specimen was fixed upon a mechanical stage in such a way that the direction of motion was vertical to the cortex. It was examined with a Zeiss apochromatic objective, 4 mm. focus, combined with the compensating eye-piece 6, tube 160 mm., thus giving an enlargement of 375 diameters.

The eye-piece carried the micrometer with 50 divisions. With the objective used, each division had a value of 4 μ. The whole scale covered therefore 50 times .004 mm. = .2 mm.

Placing the micrometer scale so that it was at right angles to the direction of motion for the specimen, and passing the specimen in review by means of the mechanical stage, a strip of cortex .2 mm. wide could be brought, throughout its entire extent, under the scale. In this manner the nerve cells were sifted, so to speak, through the micrometer scale, and each one that was 12 μ or more in diameter was picked out and counted.

In selecting the point on the section at which to make this test I always took the spot where the cells were apparently—to a low power—most abundant, and in all cases everything in the field that could be counted was counted.

The depth of the cortex where the count was made was multiplied by the constant width, .2 mm., and the total number of cells divided by this product, using .01 sq. mm. as the unit. The thickness of the section was always .02 mm., which being a constant factor may be neglected. By this treatment it comes out that about one cell, 12 μ or more in basal diameter, normally occurs in each .01 sq. mm. of a section .02 mm. thick.

For comparison with the Bridgman sections I took those from Control III. (Brain weight 1,393 gr., male, average thickness of cortex R. H. 2.77 m., L. H. 2.86 m.), and Control XI. (Brain weight 1,196 gr., female, average thickness of cortex R. H. 2.74 m., L. H. 2.74 m.), (see Table IV.), thus happening to get both the male and female with the thinnest cortex.

Table X. shows that, taking the average of both sides, at no locality in the Bridgman brain are the large nerve cells, as abundant as in the controls. The number in both the controls is nearly the same.

Taking the matter more in detail the motor areas in Laura do not show as great a poverty of large cells as the sensory areas.

In three instances (marked c in Table X.), the abundance of cells accords with the thickness of the cortex—i. e., the thicker cortex has the larger number of cells. These instances include the ones in which the Bridgman cortex most clearly deviates from the normals.

As in the measurements of cortical thickness, so in the abundance of cells, the Bridgman brain is clearly deficient at 6, the auditory area and in the right hemisphere at 11 and 14, visual area, while in the left hemisphere some deficiency is to be noted only at 14, thus again bringing out the contrast between the occipital regions on the two sides. Locality 10 has fewer cells than the controls, but the difference is not so marked as in the thickness of the cortex.

In general it may be added that where the number of cells above 12 μ. in basal diameter was small, that there the absolute number of large cells appeared smaller, and the very largest cells not so large, as in the controls. In other words, small number and small size of large cells appeared to be associated, though I have no figures to present on the point. If, however, my impression is correct, then Table X. only in part represents the difference in the development of the cortical cells of Laura as compared with the controls.

SUMMARY.

I.—General.

1. No figures can be given for the average thickness of the fresh normal cortex. The various investigators differ widely in their results. My own results agree most closely with those of Jensen.

2. Persons with an acquired defect of the central nervous system have a thinner cortex than normal persons.

3. Females have a slightly thinner cortex than males. Difference less than 1%.

4. The right hemisphere (normally) has a cortex a few per-cent less thick than the left. Maximum difference 7%.

II.—Special.

1. The cortex of Laura Bridgman was abnormally thin, having but 89% of the thickness of the controls. If we suppose that in its other dimensions the cortex was similarly reduced in development, i. e. by 11% in each linear measurement, then its normal extent would have been 246,808 sq. mm. instead of 200,202.5 sq. mm. as found. This estimate is similar to some of those by the Italian observers, Calori ([48]) and De Regibus ([17-p. 276]).

2. The right hemisphere had on the average the thinner cortex—specially to be associated with the defective visual area.

3. The thinning in the motor areas was not so well marked as in the areas for the defective senses.

4. Cortex of motor speech centre was not thin.

5. Cortex of area for dermal sensations was well developed.

6. Auditory areas (6) on both sides and visual area on right side (11, 8, 14) remarkably thin.

7. Area for taste and smell (10) thin—associated with the generally undeveloped state of the temporal lobe.

III.—Histological.

1. The cortex of Laura Bridgman contained an abnormally small number of large nerve cells—i. e., cells 12 μ. or more in transverse basal diameter.

2. There were fewer nerve cells in the samples from the right, than in those from the left hemisphere.

3. The deficiency of nerve cells was not so well marked in the motor as in the sensory areas.

4. In the centre for motor speech (2) the number of nerve cells was abnormally small.

5. Number of nerve cells very small in the auditory areas (6), both sides, and in the visual area (11, 8, 14) on the right side.

6. Some diminution in the number of cells at (10), area for taste and smell. Region generally undeveloped.

7. The small number of cells was associated with small size of the largest cells.

The persistence of vision, though in a very defective form, is still of great importance to the full development of the visual cortex—e. g., right eye and left visual area in Laura.

Observations on the Olfactory Region.

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Description of the Specimen.

The specimen submitted for examination was a portion of the ethmoid bone, extending from the anterior base of the crista galli to the sphenoid bone, a small part of the sphenoid being included in it. It contained nearly all the perpendicular plate of the ethmoid. At the sphenoidal end the lateral surfaces were devoid of mucous membrane; towards the frontal end the surfaces were quite covered with the remains of membrane in a ragged condition. The right superior turbinated bone presented a smooth surface marked with grooves. Between it and the perpendicular plate was mucous membrane. Little of the left superior turbinated bone remained, and that which did was rough and without grooves. The entire specimen measured from the extreme frontal to the sphenoidal end, 3 cm.; from the apex of the crista to the farthest point on the perpendicular plate, 2.2 cm.; laterally its greatest measurement was through the horizontal plate of the ethmoid, .5 cm. This line represented the base of two triangles; the apex of one being the tip of the crista, that of the other the farthest point on the perpendicular plate of the ethmoid.

The specimen had been hardened in Müller's fluid, and decalcified in a saturated solution of picric acid, the process being completed in a 1% solution of hydrochloric acid. It was imbedded in celloidin, and most of the sections were stained with Delafield's hæmatoxylin and eosine. Four additional stains were used for nerves, viz.: Upson's carminic acid, Schæfer's nigrosine, hæmatoxylin und carminic acid, and Pal's hæmatoxylin.

Results of the Microscopic Examination.

For the purpose of comparison, I obtained a specimen

similar to the one under consideration. This was a portion of the ethmoid bone taken from an elderly man who had been a patient at the Worcester Insane Asylum, and had died there. The presumption would be that this specimen could not be taken as a type of the normal, for it is difficult to suppose that one could pass the greater part of a long life in this climate without having had more or less nasal catarrh. The specimen was, however, healthy in its gross appearance: that is, it was symmetrical, both superior turbinated bones were present; their surfaces were shiny and grooved; the mucous membrane was generally and uniformly distributed between the perpendicular plate and the superior turbinated bones. The next point to consider was its microscopic appearance, and here arose the question, What is our standard for the normal? The work in this region has been done mainly upon the lower animals, and while the results obtained are in the main applicable to the olfactory region of the higher animals, including man, obviously it would be of great assistance to have well-conducted studies upon the olfactory region of man. In an investigation upon the olfactory region of a case of leukæmia Hermann Suchannek(84) has touched upon this topic. He has figured a microscopic section of the olfactory region of a man 40 years old, with a normal sense of smell. The picture agrees with the usual description of this region. It represents a section consisting of a regular row of epithelial cells, resting upon a basement membrane, beneath which are many Bowman's glands, a few blood vessels and nerves, with little intertubular connective tissue. Unfortunately no measurements are given, either of the entire mucous membrane or the epithelium. My specimen presented a different appearance. The epithelial layer preserved for the most part its normal characteristics of a regular row of columnar cells resting upon a row of round cells, the epithelial cells being well formed and distinct. In many places, however, the surface was not so well defined, but was breaking into crowded irregular masses of granular matter, while the subjacent layer of round cells had disappeared, and its place was taken by a mass of round cells, which penetrated deeply the underlying tissue. In these localities the surface layer of cells was thrown

into folds which projected above the surface, and also ramified into the mucous membrane, like glands. There was a general increase of connective tissue. The thickness of the entire mucous membrane varied from .16 mm. to .88 mm. Those localities that measured .16 mm., taking as a standard the usual description and the figure of Suchannek, were fairly normal. The epithelium of these regions was particularly healthy. The epithelial layer varied from 30μ to 98μ in thickness (Kölliker quoted by Schwalbe([85]) gives 40μ to 98μ as the normal thickness). It was thinnest at the extreme vault of the olfactory fissure.

In the Bridgman sections the thickness of the mucous membrane entire varied from .16 mm. to .64 mm., and the thickness of the epithelial layer from 48μ to 90μ. Taking .16 mm. as the thickness of the normal mucous membrane, I found those areas of the mucous membrane that were of this thickness, far from normal. The surface of the epithelial layer was covered with thin granular matter, and the surface line was very irregular. The cells took the stain poorly, showing that they were degenerating into mucus. In many places the cell bodies had entirely disappeared, leaving a mere outline of their former structures. The row of round cells had disappeared and its place was taken by a mass of cells, now pushing up into the epithelial layer, now invading the membrana limitans. In the sub-epithelial tissue there was a dense deposit of connective tissue. In no part of the specimen was the epithelium healthy. At some points the mucous membrane was entirely devoid of epithelial cells; at others, there was the row of round cells, now single, now two or three deep. In some places these cells were becoming polygonal in shape; again over them was a crowded confused mass of irregular cells breaking away. In some places there were breaks of continuity in the line of epithelial cells, otherwise fairly regular in their size and distribution. There were also places where the surface of the mucous membrane was thrown into elevations. There was generally a large increase of connective tissue, which, in some areas, had replaced everything else. In other areas was abundant infiltration of small, round cells. Bowman's glands were very irregularly distributed and varied

much in their character. They presented all gradations from a ring of fairly healthy polygonal cells to a confused mass of granular matter.

The mucous membrane on the right of the septum was much healthier than that on the left. Its thickness was uniform, though in some places there was an increased deposit of connective tissue. The curve into the vault of the olfactory fissure was uninterrupted and regular throughout this side of the specimen. The epithelial cells, though individually undergoing degeneration, were fairly regular in outline. Bowman's glands were numerous in the frontal part of the specimen, but toward the sphenoidal end they had disappeared. Throughout this area were nerves and blood-vessels, with greatly thickened walls. The left side of the specimen presented a very different picture. In the frontal fifth of the olfactory fissure was crowded a mass of connective tissue, in which were nerves, blood-vessels, glands, covered ventrad with degenerated epithelium. Still ventrad to this, the perpendicular plate was devoid of mucous membrane, as was also that part of the superior turbinated bone which remained; the greater part of this bone was either in small fragments or had entirely disappeared. The remaining four-fifths of this side of the specimen was occupied by a fibrous tumor, which was, as it were, in a closed cavity, the mucous membrane of the septum having firmly united with that of the superior turbinated bone, giving in the sections the appearance of a ring lined with epithelium, enclosing the tumor. The tumor sprang from the septum and projected into the superior meatus. Its length from its frontal to its sphenoidal end, estimated by the number of sections in which it was found, was upwards of 1.5 cm. It was irregularly polygonal in shape, and measured at its frontal end 1.12 mm. in height (that is, from the septum to the apex of the tumor) and 1.05 mm. in breadth, while at its sphenoidal end the corresponding measurements were 2.50 mm. and 1.44 mm. Its character changed from the frontal to the sphenoidal end. In the frontal region it was made up of a central column of dense connective tissue, which supported nerves, blood vessels, Bowman's glands, the whole being covered with a layer of epithelium as healthy as that in any part

of the specimen. At the sphenoidal end the central column was divided by a fissure, Bowman's glands had disappeared, and the whole tumor was filled with spaces of irregular shape, many of them full of blood corpuscles. Blood vessels remained, but there were few nerves and the greater part of the tumor was devoid of epithelium.

The Nerves.

There were two varieties of nerves in the specimen, a branch of the ophthalmic division of the fifth which passes into the nose through the fissure at the base of the crista galli, and the olfactory nerves. The branch of the fifth, a medullated nerve, was in the main normal. The axis cylinders stood out sharply throughout the greater part of the sections. In some areas, however, they had lost this distinctness and showed signs of beginning degeneration. But the change was no greater than might be expected in a woman of Laura's age.

Before entering upon the description of the olfactory nerves of this specimen, it will be well to discuss briefly the normal and pathological anatomy of the olfactory nerve in general.

The generally accepted view of the non-medullated nerve, of which the olfactory is a type, is that it is made up of the so-called Remak's fibres. Each of these consists of an axis cylinder, a neurilemma, and between the two a nucleated nerve corpuscle from place to place. This fibre has a striated appearance due, according to Max Schultze, to the fibrillæ of the nerve, which are distinguished from the axis cylinders of a medullated nerve in that they individually have no medullary sheath. Boveri(86), on the other hand, has made a careful study of this subject, and concludes that the fibrillæ of Max Schultze are really nerve fibres, each having a medullated sheath. This sheath does not, however, belong exclusively to each nerve. It sustains the same relation to the contiguous nerve fibres that the cell wall of a honey-comb does to the cells. A number of these nerves are surrounded by an envelope of connective tissue, in which are here and there stellate connective tissue corpuscles. There are also within the investing sheath, among the nerves, connective

tissue corpuscles, with stellate rays which can be traced very far, even to the enclosing sheath.

The olfactory nerves are subject to certain definite pathological conditions. In the first place, they may be congenitally absent. Injury to the head may cause rupture to the nerves as they pass through the cribriform plate. Excessive stimulation may temporarily or permanently destroy their excitability. Tumors in the brain or cerebral hemorrhage may by pressure cause disease of the olfactory nerves. There may be atrophy of the bulb or nerves, or they may be affected by the degenerative changes of old age. Simple neuritis is a very rare affection (Althaus) ([87]). Chronic neuritis, due to syphilis, however, is not uncommon. The nerve may become involved in local inflammatory changes in connection with meningitis. Bosworth([88]) is of the opinion that a very frequent cause of anosmia from diseases of the olfactory nerves is due to the influence of local inflammatory changes. Thus in acute rhinitis, anosmia persists many days after the inflammatory process undergoes resolution. In severer disease of the nose, where the local inflammatory action persists longer, or is of a severer type, the anosmia lasts much longer, long after the inflammatory action has subsided.

To return to our specimen. The nerves were numerous and were easily distinguished by moderate powers of the microscope (320 diameters.) To get some definite idea of the distribution of the nerves in the different parts of the specimen I selected five slides and counted the nerves on them. One of these sections was from the frontal end, one from the sphenoidal, the other three at regular intervals between them. I also made a count of the nerves of the control specimen under similar conditions, with the following results, (The slide numbered one in each case, was from the frontal end).

Bridgman.		Control.	
1st slide,	1 nerve.	1st slide,	4 nerves.
2nd "	7 "	2nd "	20 "
3rd "	28 "	3rd "	10 "
4th "	32 "	4th "	8 "
5th "	18 "	5th "	15 "
Total	86	Total	57

This enumeration is of interest in that it shows the distribution of the nerves in the different parts of the specimens, but it gives no reliable information as to the relative number of nerves in the two specimens. It is a difficult matter, even under favorable conditions, to stain the olfactory nerves so as to show the nerve fibres. In neither of these specimens was I able to show the olfactory nerves with the special stains for nerve tissue. The only stain that brought them out at all was the hæmatoxylin and eosine, which did it by virtue of its differentiating the connective tissue. We shall see that this latter was greatly increased in the Bridgman specimen, and it is evident that because of this many more nerves would be detected than in the healthier specimen.

In the Bridgman slides, the nerves were surrounded by a ring of connective tissue which was very thick. Within this ring was a uniformly granular field broken up into smaller areas, and more or less studded with deeply stained dots. With a $\frac{1}{12}$ oil immersion objective, these dots were seen to be stellate connective tissue corpuscles. The areas alluded to above corresponded to the portions of the nerve bounded by the connective tissue envelope in Boveri's sections. Here, as with his sections, the connective tissue corpuscles were upon and within the sheaths. Rather the connective tissue corpuscles of the sheath were where the sheath should be, that place being represented in our sections by a vacant space. As this apparent shrinking was quite general throughout the specimen, I attributed it to the action of reagents. With this power, the nerve presented a regularly mottled appearance, very similar to a section of a frog's olfactory nerve as figured by Boveri, and representing according to his views the cut ends of the nerve fibres. The nerve in its essential elements, therefore, was normal. The connective tissue elements, however, were largely increased.

General Considerations.

It will be interesting now, to gather together the available facts relating to Laura's sense of smell, and the general condition of her nasal mucous membrane during life, and to find, if we can, in the condition of this membrane an explanation of her symptoms.

As an infant she was delicate, being subject to severe convulsions. But later her health improved, and when two years old she is described as being more active and intelligent than ordinary children. At two she had scarlet fever with such severity that for seven weeks she was unable to swallow solid food. Both eyes and ears were affected, suppurating freely ([1–p 2]). When seven years old she was seen by Dr. R. D. Mussey, Professor of Anatomy and Surgery at Dartmouth College, and in a letter dated April 14, 1837, he thus alludes to her sense of smell: "Her sense of smell is thought by her mother to be less acute than other children, as she very seldom applies any odorous substance to her nose: it is not improbable that this sense may have been impaired by the fever"([1–VIII]). In this year, 1837, she entered the Perkins Institution and we find in Dr. Howe's report this note ([2-155]): "For all purposes of use she is without smell, and takes no notice of the odor of a rose, or the smell of cologne water, when held quite near her, though acrid and pungent odors seem to affect the olfactory nerve." April 6, 1842, Miss Swift, Laura's teacher, made this note([1–p. 107]): "Dr. Howe came into the room, while she was having a lesson, peeling an orange. She stopped in the midst of a sentence to say, 'I smell an orange.' We can see a decided improvement in her sense of smell since last year, but she has never noticed any perfume so quickly or at so great a distance before." June 19, 1844, we find this note([1–p. 257]): "This is the first season she has ever perceived the smell of a rose or pink, and she now puts all flowers to her nose and is disappointed if they have no perfume. In a letter to Mrs. Howe, dated June 25, 1844, Laura herself says([1–p. 258]), "I can smell roses much better than I did two years ago, and it gives me much pleasure in smelling roses."

I find but few observations upon the general condition of her nose. Dec. 14, 1843, Miss Swift made this note([1–p. 215]): "She has always been a sufferer from a severe catarrhal affection, and as this shows signs of improvement, we hope for a corresponding one in both smell and taste." In 1878 Dr. G. Stanley Hall, in the course of a series of observations upon her several faculties, examined her nose with this result([6]):

"There is no deformity or scarification observable without or from a cursory examination within the nose, and the yellow pigment of the Schneiderian membrane can be seen by a very simple apparatus." Dr. Hall made further this very interesting observation. He described her sleeping with long regular breathing, the teeth slightly apart and the tongue pressed against them and almost between them.

I have received the following letter from Miss Della Bennett, who has been a teacher in the Perkins Institution since 1876:

"Laura Bridgman lived for several years in the same family with myself, and I have conferred with the matron of the cottage, and can answer most of your questions definitely. There was copious discharge from her nose, so much so that she was wont to say, 'My poor nose!' Her handkerchief was in frequent demand, and she used many. Her breath was never offensive. She always breathed through her nose, a habit which she formed when quite young, and her breathing was often accompanied with a gentle whistling sound. I have seen her asleep in the daytime and her mouth was closed, but I cannot tell about the night. She did remove mucus from her throat, and occasionally had a sore throat."

From these notes one gathers that at the age of two, Laura suffered from a severe inflammation of the naso-pharynx, which doubtless extended to her nose: that after her illness she was quite destitute of the sense of smell, entirely so when at the age of eight she entered the Perkins Institution: that at the age of fifteen she could detect with certainty and pleasure moderately pronounced odors: that she had a severe nasal catarrh which lasted her entire life, although it decreased somewhat in severity: furthermore that there was no deformity without or within the nose that could be seen by one not accustomed to examine these parts.

We now come to the consideration of the cause of Laura's anosmia and her partial recovery from it. We have seen that the olfactory nerves were capable of performing their function, and according to Dr. Donaldson (*vide ante*) there was no central lesion that would cause anosmia. We must therefore seek for the cause in the periphery of the nervous

apparatus. The two chief peripheral causes of anosmia are obstruction to the inspired air due to deformity of the nose, hypertrophy of the turbinated bodies, nasal polypi or tumors, and atrophic disease. That there was not atrophic disease is shown by the absence of bad odor, by the partial return of the sense of smell, and by the result of our examination of the specimen. Furthermore, according to Bosworth, catarrhal affections caused by febrile diseases and prominently scarlet fever, are characterized by hypertrophic changes ([88—p. 157]). It is quite improbable that Laura had any deformity of the nose or hypertrophic disease in the respiratory part of the nose, which would interfere very materially with the access of the inspired air to the olfactory region, and it is in this latter region, therefore, that we must look for the cause of her anosmia. . We have found in the left superior meatus an adequate cause for a complete absence of the sense of smell for that area, in the extensive disease there which resulted in a thorough disorganization of the mucous membrane in a part of the olfactory fissure, while the rest was excluded from all contact with the inspired air by the firm union of the mucous membrane of the septum with that of the left superior turbinated body. In the right superior meatus, on the other hand, conditions were more favorable for the proper performance of function. It is here that Laura must have smelled, and the questions now to be settled are, how could this area have been rendered incapable of performing its function, and how could this function have been resumed.

Catarrhal inflammation of the nasal mucous membrane is the usual accompaniment of scarlet fever, except in the mildest cases, and is associated with an irritating discharge from the nose (Smith)([89]). The inflammatory process in these cases does not involve more than the epithelial layers. But in severe disease the deeper tissues of the mucous membrane are affected. There is a copious proliferation of cells in the deeper layers, with fibrinous infiltration even to the extent of compressing the vessels and making portions of the tissue gangrenous (Henock)([90]). There may even result necrosis of the bones (Thomas)([91]). There may be recovery even though the disease be severe, or it may result in chronic disease with

more or less profuse discharge and extensive inflammatory infiltration, or there may be an osteitis of all the bones which enter into the composition of the nasal cavities (Allen)([92]).

That Laura's nasal mucous membrane was profoundly affected by the fever there seems no doubt, and it is easy to conceive how the active cell proliferation and swelling of the mucous membrane caused by the catarrhal process would have so affected the delicate termination of the olfactory nerves that they would be entirely incapable of functioning. But as time went on we know her catarrh grew better and we rightfully infer that the inflammatory processes in the mucous membrane subsided, to an extent, though they never entirely ceased. We have seen that the structures of the nose were a good deal damaged, yet they were not entirely useless. In the right superior meatus especially, there were spots of membrane in a fairly healthy condition. A question of interest here presents itself—would the olfactory nerves after so long a period of inactivity preserve their power of responding to stimuli? The following case reported by Allen ([92]) proves that this is possible. The patient was a married woman. She had never breathed through her nose and had never experienced the perception of an odor. There was found to be a complete bony occlusion of the posterior nares. This was broken through and on the sixth day after the operation she began to smell and in a short time became familiar with the common odors and flavors. The odoriferous air was not kept from Laura's olfactory nerves by bony obstruction, but it was kept from them by what acted as efficiently for a long time, namely, masses of rapidly proliferating cells, and the mucus and débris of a diseased mucous membrane. When this process subsided it again became possible, in those areas where the ephithelium still remained sufficiently healthy, as it did in places, for the terminal filaments of the nerves to receive and convey their proper stimuli. There may have been a further cause for the anosmia. When discussing the pathology of the olfactory nerve, we alluded to Bosworth's view that anosmia was due in some cases to the local action of the surrounding inflammation upon the nerve itself. As I understand the matter he bases this view solely upon clinical experience, and attempts no explanation of the tardy return of the sense of

smell after the subsidence of the inflammation. We have in our sections a possible explanation of this peculiarity. The connective tissue of the nerve was increased in amount, while the nerve tissue proper was apparently normal. Interesting questions suggest themselves in this connection. Does the development of this tissue impair the functioning power of the nerve, and does a nerve so affected resume its normal activity more slowly than the surrounding tissue? At present, so far as I know, there is not sufficient anatomical data upon which one could even discuss these topics.

Summary.

I. The ethmoid bone and the mucous membrane covering it had suffered from inflammatory disease, which particularly affected the left side. 2. This disease resulted in an excessive production of connective tissue, and in one area, the left superior meatus, there had been formed a fibrous tumor. The epithelium was generally and considerably diseased. The nerves contained an excess of connective tissue, but were otherwise normal. 3. When two years old, Laura had scarlet fever, which left her anosmic and with severe nasal catarrh. She partially recovered from both these conditions. 4. The anosmia was due to the occlusion of the left olfactory area by the union of the mucous membrane of the septum with that of the superior turbinated body, and also to the action of the inflamed mucous membrane upon the nerves of the right olfactory region. Partial recovery resulted from subsidence of this inflammation.

II.—The Visual Apparatus.

When Laura recovered from her illness it appeared that she was totally blind in her left eye but could see somewhat with the right. The remnant of vision in her right eye continued up to the eigthth year of her life.

From that time on she was absolutely blind in both eyes.

In 1878 Dr. O. F. Wadsworth, of Boston, tested her for vision and found her totally blind (6) and at the same time reported on the appearance of the eyes as follows:

"On both sides the lids are sunken, partly on account of lack of the normal amount of orbital fatty tissue, partly on account of the small size of the eyeballs. They remain constantly closed. The right conjunctival sac is much smaller

than normal, somewhat irregular, and presents an appearance such as is seen after severe and long-continued inflammation. The right eye appears about one half the normal size. It is wholly enclosed by the sclerotic, except over a space at the centre, some two millimetres in diameter, where a less opaque tissue, on which a few blood-vessels are visible, represents the altered remnant of the cornea. The left conjuctival sac is somewhat larger than the right, and more regular, though still small. The left globe also is a little larger than the right, and its opaque altered cornea is some four millimetres in horizontal and two millimetres in vertical diameter. There was constant irregular oscillation of the globes [nystagmus] whenever they were exposed to view by raising the lids, and the oscillation evidently continued even after the lids were closed."

At the autopsy the eyes were removed with the surrounding tissue and put unopened into the Müller's fluid and alcohol. The hardening was completed in alcohol.

Both bulbs were enclosed by orbital fat. All the muscles the of bulbs were present, though small, and the external appearance of the bulbs corresponded with Dr. Wadsworth's description given in 1878. After hardening, the right eye had a transverse diameter of 15 mm. and an antero-posterior diameter of 10.5 mm. Similar measurements of the left eye gave 17.5 and 11. mm. showing the left to be decidedly the larger. The condition of phthisis bulbi existed for both eyes. There was a faint indication of the anterior chamber. The locality of lens and vitreous contained abundant calcareous deposits in small masses and the choroidal pigment was very abundant. Sections through the point of entrance of the optic nerve showed no trace of the retina or normal nervous elements at this point. Both eyes were similar in the appearance just mentioned. As has been stated the optic nerves were small:

Right optic nerve, area of cross-section near chiasma, 5.00 sq. mm.
Left " " " " " " 3.38 " "

The connective tissue was vastly increased in both nerves but one also saw the characteristic cross sections of axis-cylinders with their medullary sheaths. The fibres were both

large and small. It is worth noting that these fibres were abundant in the left nerve but much less so in the right, although the right was the larger nerve. The chiasma was much flattened dorso-ventrally. The optic tracts were small and flattened. Their area was taken about 10. mm. behind the chiasma. The relations of size were of course reversed at this point and the left tract was the larger:

Right optic tract near chiasma,	3.13 sq. mm.
Left " " " "	4.69 " "

From these measurements the only conclusion that can be drawn is that a large part of the fibres decussated. In the tracts, which were not very well hardened, the fibres visible in cross-section of the corresponding optic nerves were also to be found. Throughout the nerves and tracts, but more abundant in the latter, there were numerous droplets or spherical homogeneous masses, as a rule about 12 μ in diameter, and staining with fuchsin and carmine. Lying at the periphery of both nerves and tracts these bodies would appear to correspond with corpora amylacea, with some of the descriptions of which, however, they do not exactly agree. Further than the tracts it was not practicable to carry the histological examination of the optic pathway.

The corpora geniculata externa were too imperfect for description. The pulvinar and the anterior pair of the corpora quadrigemina were both slightly less prominent than in the normal brains. The cortex was the next locality studied and the results there obtained have already been given.

The first point calling for remark is that the eye in which vision was longest retained ultimately had the smaller bulb and at the same time it was associated with the larger optic nerve and tract. The nerve and tract, however, though larger showed fewer nerve fibres that were clearly marked. It should perhaps be noticed in this connection that this smaller bulb had also the smaller (right) oculo-motor nerve in connection with it.

From these facts it would appear that although in general the right eye was more seriously affected yet some portion of the retina remained undamaged for a long time—up to the

eighth year. During this period the optic nerve, the tract and the cortex underwent considerable development so that the subsequent degeneration of the right nerve was accompanied by far less atrophy than that of the left side. On the left side the disturbance in the eyeball was in general less severe and though vision was abolished very early, there was left some condition which favored the better preservation of those nerve fibres which did not at an early period undergo degeneration and absorption. I had expected to find complete degeneration of both optic nerves such as had been described by Purtscher. ([80])

On the bases of these specimens, I should hardly like to enter into the forms of degeneration possible to the optic nerves but if a double set of fibres in the optic—the two sets developing and conducting in opposite directions—be accepted, then these nerves found intact in this case might be considered as belonging to that set the centre for which was central and which conducted peripherally. v. Monakow ([81, 82])

In this instance then the disturbance in the cortex is probably to be looked upon much more as due to an arrest of growth following the removal of the normal stimuli, than to a continuation of the degeneration into the hemispheres.

III.—The Auditory Apparatus.

From the time of her illness to her death there is good evidence that Laura was entirely deaf. At the same time she had a good sense of direction and of equilibrium and was sensitive to rotation. Hall ([6]). The equilibrium and auditory functions of the eighth nerve are therefore to be separated in this case.

An examination of the ears was made in 1878 by Dr. Clarence J. Blake who reported as follows: ([6])

"Both external ears normal. The right external auditory canal normal in size and contour, and the skin lining the passage healthy and showing no marks of previous inflammation-processes. The right membrana tympani was entirely destroyed with the exception of a narrow rim, the remains of the inferior and posterior portions of the membrane, from which a thin cicatrical tissue extended inward to the promontorium over the stapes and fenestra rotunda. The malleus

and incus had disappeared. The mucous membrane of the tympanic cavity presented a normal appearance, with the exception of one spot on the promontorium covered with a thin crust of dried secretion about two millimetres in diameter. A band of thin cicatrical tissue also extended across the anterior portion of the tympanic cavity. The left external auditory canal was filled with dark brownish cerumen, on removal of which the passage was found to terminate, at a depth of two centimetres, in a diaphragm of secondary granulation-tissue, concave, very firm, and resisting gentle pressure with a probe, except at the central or thinner portions, where it could be slightly depressed. Its outer covering was continuous with the dermoid lining of the canal."

After death, the petrous bones were put in Dr. Blake's hands and the report on them, made by Dr. W. S. Bryant, of Boston, is the following:

The Examination of Laura Bridgman's Petrous Bones.

The Right Petrous Bone.

A deep groove for the superior petrosal sinus is seen. The external auditory canal is terminated by a concave curtain of fibrous tissue resting on the promontory. There is no evidence left of the former position of membrana tympani except at the floor of the canal, where there is a slight indication of the sulcus tympanicus. The tympanic cavity is considerably constricted by hyperostoses. The oval and round windows are ossified across and the promontory is very rough, leaving only a small space inferiorly and posteriorly. The inferior anterior wall of the tympanum is very thin and there are two pin-hole perforations into the carotid canal.

The Eustachian tube is impervious; its tympanic end being closed by bone and just beyond this there is an accumulation of cheesy matter also enclosed by bone. There are no air spaces within the tympanum for all the bone cells are filled with tissue, although in the highest part of the petrous bone there is a cell which connects with the tympanum. There is no evidence of mastoid cells or antrum. (I did not see the mastoid process).

The chorda tympani muscle is very much atrophied and its tendon is attached to cicatricial tissue. The stapedius

was very much atrophied and its canal narrowed. The tendon still protrudes from the tubercle.

Anteriorly and externally the osseous wall of the aqueduct of Fallopius is wanting. No trace of the ossicles could be found. The inner ear appears normal.

Dr. H. F. Sears kindly examined the terminations of the auditory nerve and organ of Corti and found the terminal ganglion cells intact.

The Left Petrous Bone.

The groove for the superior petrosal sinus is unusually deep. A diaphragm of dense fibrous tissue especially thick and firm on the surface and concave outwards forms the end of the conical external auditory meatus 8 mm. external to the base of the styloid process.

The floor of the osseous meatus is defective externally and is pierced internally and anteriorly by a foramen 1 mm. in diameter, in the fissure of Glacier.

External to the fibrous diaphragm there is a diaphragm formed by hyperostosis of the walls of the canal which obstructs the passage except near the centre and slightly external to the normal position of the membrana tympani, where there is an opening 2 x 4 mm.

The hyperostosis extends into the tympanum filling the greater part of it, but leaving a space external to the fenestræ and below the promontory, also a considerable space in the external anterior and superior part of the petrous bone.

There are no air spaces between the place of closure of the meatus and the pharyngeal end of the osseous Eustachian tube. All the bone cells are filled with soft tissue and the osseous Eustachian tube is not seen. No remains of the membrana tympani could be found.

Before I saw the specimen the tympanum had been opened and some of its contents taken out; all of this was lost except the head and neck of the malleus with the base of the long process, all enclosed in fibrous tissue.

The relations of the fenestra ovalis and the attachment of the tensor tympani muscle had also been destroyed. The chorda tympani nerve was found intact. The tendon of the stapedius muscle was protruding from its tubercle.

The aqueduct of Fallopius and its contents are intact. The round window is closed by dense fibrous tissue. Both the round and oval windows are small, less than one-half of normal size.

Dr. H. F. Sears kindly examined the nerves and muscles and found the tensor tympani considerably and the stapedius slightly atrophied. He also found numerous ganglion cells in the cochlea.

The original report of Dr. Bryant ends here. In answer to a further question, however, he states that nothing pathological could be definitely made out in either the cochleas or semi-circular canals. As the original preservation of the specimens had been in Müller's fluid only, they were not in the best condition for a fine histological examination.

As the case stands the inflammation of the middle ear is the occasion of the deafness. The authorities on the subject state that absolute deafness does not follow disease of the middle ear alone. So that there is something here to be explained. I consider that the cochlea must have been thrown out of function on both sides since the tuning fork placed on the skull gave no auditory sensations—and this, to my mind, outbalances the negative result of the histological examination.

The auditory nerves were studied only by means of the stumps attached to the medulla; the right auditory had an area of 4.26 sq. mm. in cross section. The left of 3.17 sq. mm. Both samples were taken within about 3 mm. of their attachment to the medulla. (For the method see the article in this same number on "The size of several cranial nerves in man as indicated by their cross-section.") Roughly their area was about two thirds of that of the similar nerves from the brain of a normal male in whom the cranial nerves were all very large. There is no reason then to think that in Laura the nerves were remarkably small. The figure for the area of the larger, right nerve, is somewhat too high owing to the obliquity of section and some distortion, so that they were really more nearly equal than these figures would indicate.

The connective tissue in the nerve trunks is normal. The

nerve fibre show well marked sheaths and axis cylinders. If degeneration has occurred in these nerves the indications of it have long since disappeared. The nerve fibres found would be designated as normal. The bundles of larger fibres, presumptively connected with the semi-circular canals, contain particularly well preserved fibres.

In the medulla both roots and all three nuclei can be clearly identified on both sides.

The fibres in the medulla stain by Weigert's method and the cells with carmine, as well as could be expected from the condition of the specimen. If there is any abnormality it is that the auditory fibres do not take the Weigert's stain particularly well and that the cells of the accessory nucleus in the medulla are few and poorly developed. The striae acusticae were well developed and on gross examination—when the floor of the fourth ventricle was viewed from above—there were visible two bundles on the right side and three on the left which could be counted as belonging to the striae, while just cephalad to these was a well marked bundle on each side of the middle line, corresponding with the structure described as the *conductor sonorus* (Klangstab) and supposed to form part of the centripetal pathway for the auditory impulses.

On comparison with a number of normal specimens the caudal pair of the quadrigemina exhibited no marked peculiarity. They were small, but no smaller than in the case of some normals. The corpora geniculata interna did not appear small in Laura upon gross examination but this appearance I am inclined to attribute to the failure of the surrounding regions to fully develop, thus causing the corp. gen. int. to stand out with unusual clearness.

The next point examined in the auditory pathway was the cerebral cortex and the results there found have already been stated.

I wish to add in this place that in the description of the surface of the brain previously given Iwas not willing to admit any superficial abnormality in the region of the first temporal gyrus at its caudal end. Since writing that description I have made further comparisons with normal brains and have obtained evidence of lack of development in the cortex of this

region in the case of Laura. At present then I look on the slenderness of this gyrus, especially on the right side, where the cortex is most affected, as an expression of the incomplete development of the region. Mills ([42,68]), Starr ([41]), Manouvrier ([83]).

At first sight the small disturbance—to the naked eye at least—existing between the middle ear and the cortex is striking. Histological investigation up to the centres in the medulla yields a similar negative result. Between the medulla and cortex the condition of the specimen did not warrant a histological study.

In the scattered literature relating to the examination of the ear and brain in deaf-mutes, a condition where there is little or no apparent abnormality of the inner ear, the auditory nerve or the medulla, associated with disease of the middle ear, deafness and (sometimes) atrophy of the cortical auditory centres, is occasionally described: Bremer ([69]), Larsen & Mygind ([70]), Moos ([71]), Mygind ([72]), Obersteiner ([73]), Moos and Steinbrügge ([74,75,76]). I believe that in future cases, like that of Laura, a more detailed examination than it was possible to make in her case will show disease of the membraneous cochlea or the nerves between it and the spiral ganglion of the cochlea. Such a case has been reported by Moos and Steinbrügge ([79]).

As long, of course, as the cells of the spiral ganglion are intact, just so long will the auditory fibres associated with them—and this must represent a very large portion of the cochlear division of the auditory—remain morphologically intact. Following the pathway to the cortex we find no point at which marked changes occur until we reach the cortex itself. The disturbance here is most probably due to the early and long continued lack of normal excitation, for the cortical cells in the sensory areas are peculiarly dependent for their proper development on the special sense with which they are associated.

The evidence from stimulation of the cortex and from the histology of the medulla goes to show that the association between the auditory nerve and the cortical centre for hearing is to some extent at least, a crossed one. If this were so, then

the smaller, left nerve, would associate itself with the thinner, right cortex. This relation exists in the case of Laura, but it remains for further investigation to show its significance. Strümpell ([78]).

As regards the semicircular canals it may be added that they were not found diseased. Their nerve was in good condition, and sensibility to rotation, sense of direction, etc., were present. Of course the relation of this part of the inner ear to the middle ear is less intimate than that of the cochlea, and this in part may account for the normal preservation of the canals. That both portions of the labyrinth need not be conjointly affected is shown by James ([79]), in his study of the sense of dizziness in deaf-mutes, where this sense was found totally lacking in only 186 out of the 519 cases examined.

IV.—The Cranial Nerves.

It is desirable to bring together the various facts regarding the cranial nerves in Laura's case. After what has been said in the foregoing pages, and the discussion of their area by Mr. Bolton and myself (vide p. 224), this can be briefly done. Table XI. gives the various points in a condensed form.

TABLE XI.

Nerve.		Area in sq. mm.	Condition.	Size.
I. Olfactory, bulb,	right	6.34	Somewhat atrophiad	Small
" " tract,	right	1.46	" "	"
II. Optic nerve,	right	5.00	Greatly atrophied	Very small.
" " "	left	3.38	" "	" "
" " tract,	right	3.13	" "	" "
" " "	left	4.69	" "	" "
III. Oculomotor,	right	3.17	Normal	Large
" "	left	3.51	"	"
VIII. Auditory,	right	4.26	Somewhat atrophied	Small
" "	left	3.17	" "	"

The sixth nerve—abducens—contained only normal fibres and appeared healthy, but the measurements on the two sides were so different that I suspect some strands were lost, and hence do not give the figures for the area.

The only nerve in the Table which has not been discussed is the olfactory. The bulb was flattened and the glomeruli could not be identified. The ganglion cell layer was there, and contained some well formed cells. The other layers were poorly preserved. The vessel walls were thickened. There was some excess of connective tissue and an abundance of

hyaline bodies—corpora amylacea (?). Distinctly degenerated fibres could not be made out in the tract, but the vessels, connective tissue, corpora amylacea, were found as in the bulb. Grossly the left tract and bulbs were like the right, but by accident the former was lost before it had been examined histologically.

Whether there was anything peculiar in the glossopharyngeal fibres I am unable to say. The portion within the medulla was normal.

The medulla which was examined from the level of the pyramid to the middle of the pons, by means of sections, showed no abnormality save in the neighborhood of the accessory nucleus of the auditory nerve, where the cells appeared small, reduced in numbers and highly pigmented.

The pia of the hemispheres had a normal abundance of nuclei in it, even over the occipital region—and the blood vessels were normal in size and thickness of their walls. The cerebellum was also normal.

V.— Conclusion.

From these fragmentary observations, which leave so many points connected with this special case still undecided, it will be advantageous to construct some sort of general picture.

The anatomical condition was that of a normal brain in which the olfactory bulbs and nerves, the optic nerves, the auditory nerves, and possibly the glossopharyngeal, had all been more or less destroyed at their peripheral ends. This destruction caused a degeneration—most marked in the optic nerves—which extended towards the centres and involved them indirectly. This condition has left its mark more or less plainly on the whole brain, as indicated by the extent and thickness of the cerebral cortex, and specially by the cortex connected with these deficient sensory nerves. The physiological effect of the peripheral lesions, as I conceive it, was to retard growth in the centres, cortical and subcortical, which were thus involved, and also to interfere with, if not entirely prevent, the formation of the association tracts.

To be sure this case represents a maximum loss in these defective senses with a minimum amount of central disturbance, thus offering the very best sort of opportunity for education by way of the surviving senses. At the same time, we must

imagine the hemispheres to have been traversed in every direction by partly or completely closed pathways. The brain was simpler than that of a normal person, and Laura was shut off from those cross-references between her several senses, which usually so facilitate the acquisition of information and the process of thought. Mental association was for her limited to various phases of the dermal sensations and the minor and imperfect senses of taste and smell. Yet from their fundamental and protean character, the dermal senses are perhaps the only ones on which alone the intellect could have lived. We are thus brought back to Sanford's (7) conclusion as derived from the study of her writings. "She was eccentric, not defective. She lacked certain data of thought, but not, in a very marked way, the power to use what data she had."

One word more upon the cortex. The deficiency in the motor speech centre is mainly macroscopic, as far as the third frontal gyrus is concerned. The motor centre there had lost some, but not all its associative connections. Histologically, it was slightly deficient. The lesion there was so different from that of the sensory centres that a histological difference ought not, perhaps, to be surprising. The cortex of the sensory centres was not sunken below the surrounding level, though the gyri were slender and flattened. Possibly in this sinking in a motor area and the absence of the same in the sensory areas, we have a suggestive difference in the reactions of the several portions of the cortex.

Finally, the deficiency was not so very great even in those areas, where it was most marked, and the question arises as to what sort of occupation the cells in those areas had, which would thus justify their prolonged existence. If they were thrown entirely out of function it is not easy to see how they could last so well for nearly sixty years. In some way then they may have taken a slight part in the cerebral activity, but it was so slight that their specific reactions did not rise into consciousness, for though Laura had some light perception up to her eighth year, she apparently had no visual memories, whereas those who have retained full vision up to four and a half or five years of age and then become blind, do usually remember in terms of sight (8).

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CORRIGENDA. I. ARTICLE.

Page 304. The percentage increase in volume is certainly too large. It should be one or two per cent. less than that for weight.

Page 306. Line 8. All the specimens mentioned in this paragraph except the Bridgman, are supposed to have been weighed with the pia on. To make this specimen comparable then its weight must be increased by the weight of the pia, 31.4 grms. This makes the total weight of the Bridgman encephalon, with pia, 1235.4 grms.

Page 312. Line 13. Topinard's Table (Éléments d'Anthropologie générale, Paris, 1885) in his Anthropology, p. 518 shows the relations between brain weight and age. It is based on 1913 cases of Boyd, and according to it the maximum encephalic weight for females, falls between the ages of 20-30 years; that for males between 30-40 years; This indicates brain growth up to the age of maximum weight, therefore beyond the twenty-fifth year.

Page 324. Table. The first series of weights stands under the heading "Weight of cerebral hemispheres, fresh." The question arises whether "cerebral hemisphere" should not be replaced by "encephala." I have not seen any account of how much of the encephalon was used in determining the fresh weights in this series, but, since these brains were directly compared with those of other observers in which the entire encephalon had been weighed, it is only fair to suppose that they had been treated in the same way. This was my opinion until I found a table in R. Wagner's[31] Vorstudien, 2te Abhandlung, 1862, P. 91, in which the weights of the two "hemispheres," of at least three of these brains in the table, are compared with one another. The specimens had been in alcohol the strength of which is not given. Now the sum of the weights of the two "hemispheres" is nearly equal to or more than the weight of the "brains" given by H. Wagner [44] 1864. I therefore used the word "hemisphere" in the above heading as equivalent to hemierebrum. It would appear that both the Wagners used it as equal to hemiencephalon. In the above mentioned table then the weights given are these for the entire encephalon and not for the cerebrum only.

Page 328.	Table I.	*For* absolute difference,	1398.4 sq. mm.
		Read " "	1598.4 " "
		For in percentage,	1.8%
		Read "	1.9%
Page 334.	Table VIII.	*For* total (left),	101256.0 sq. mm.
		Read " "	101255.2 " "
		For absolute difference,	2309.5 " "
		Read " "	2308.7 " "

PLATE III.

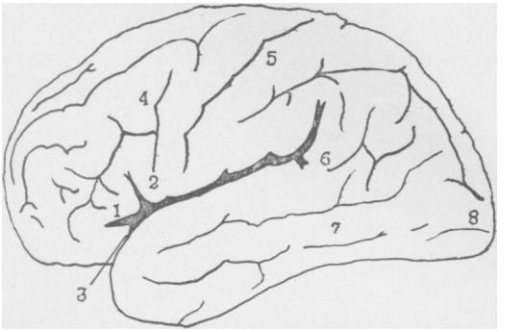

FIG. 1. Lateral aspect. 3 is used to designate the insula here not exposed.

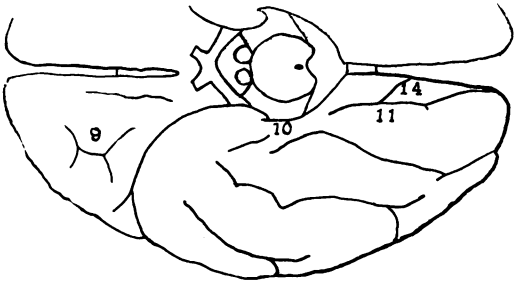

FIG. 2. Ventral aspect.

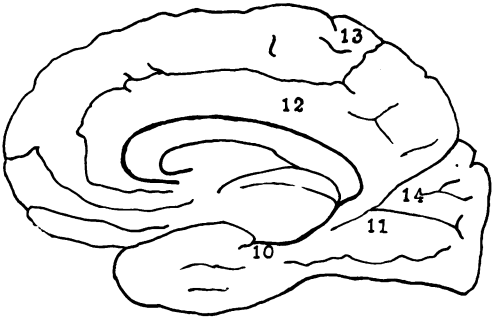

FIG. 3. Mesal aspect.

Explanation of Plate III. This plate shows the localities on the hemispheres from which the samples of cortex were taken. For the physiological value of these localities Table III may be consulted.